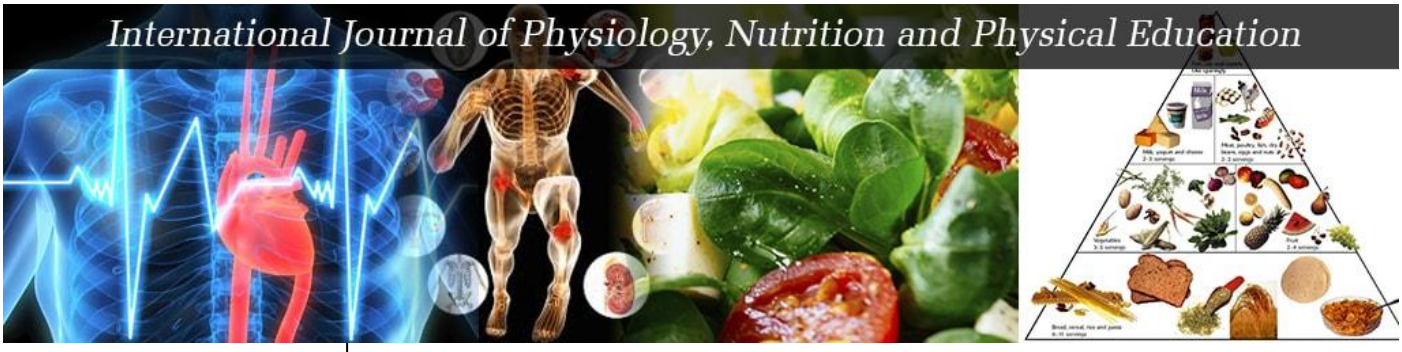

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Nerve conduction: A review

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Abstract
Neurons communicate with other cells by the release of chemical neurotransmitters. The human nervous system has some 100 billion neurons, each of which communicates with postsynaptic targets via chemical neurotransmission. The first neurotransmitters described were acetylcholine (ACh) and norepinephrine (NE). These were identified at synapses in the PNS. Many others transmitters have been identified since then, but, even counting all the peptides known to act as transmitters, the number is well less than 50. The specific neuronal signalling that allows the enormous complexity of function in the nervous system is largely a result of the specificity of neuronal connections made during development and the distribution of specific classes of neurotransmitter receptors. Nerve conduction study is done to assess whether a nerve which has suffered compression or injury is degenerating or not. The nerve is stimulated directly by a short duration stimulus along its course. When it is stimulated it conveys impulses to the muscles it supplies and the muscles contract (Downie, 1992) The principal use of nerve conduction studies is to identify damage to peripheral nerves, and to determine whether the pathological process is focal or diffuse and whether the damage is principally axonal or demyelinating. It is also possible to obtain some information about nerve roots by more sophisticated analysis of responses to impulses initially conducted antidromically to the spinal cord, and then orthodromically to the stimulation point (F wave). (Walker *et al*., 2013). In this paper, an attempt has been made to explain the neuron, neural transmission and nervce conduction study mechanism principle and measurement.

Keywords: Nerve conduction study, NCV, neural-transmission

Introduction

Neurons

Neuron or nerve cell is the structural and functional unit of nervous system. Neuron is similar to any other cell in the body, having nucleus and all the organelles in cytoplasm. However, it is different from other cells by two ways:

1. Neuron has branches or processes called axons and dendrites.
2. Neuron does not have centrosome. So, it cannot undergo division like muscle cells. (Sembulingam, 2012) [6]

Neurons (nerve cells) possess electrical excitability, the ability to respond to a stimulus and convert it into an action potential. A stimulus is any change in the environment that is strong enough to initiate an action potential. An action potential (nerve impulse) is an electrical signal that propagates (travels) along the surface of the membrane of a neuron. It begins and travels due to the movement of ions (such as sodium and potassium) between interstitial fluid and the inside of a neuron through specific ion channels in its plasma membrane. Once begun, a nerve impulse travels rapidly and at a constant strength. Some neurons are tiny and propagate impulses over a short distance (less than 1 mm) within the CNS. Others are the longest cells in the body. The neurons that enable you to wiggle your toes, for example, extend from the lumbar region of your spinal cord (just above waist level) to the muscles in your foot. Some neurons are even longer. Those that allow you to feel a feather tickling your toes stretch all the way from your foot to the lower portion of your brain. Nerve impulses travel these great distances at speeds ranging from 0.5 to 130 meters per second (1 to 290 miles/hr). (Tortora & Derrick son, 2012) [7]

Chemical Composition of a Nerve Tissue

In the adult brain, where gray and white matter are mixed, the water content averages 78%, in the spinal cord, water content is slightly less, about 75%. The solids of nerve tissue consist of mainly proteins and lipids. There are also smaller amounts of organic extractives and of inorganic salts (1%).

Proteins of nerve tissue: They constitute 38-40% of total solids, including various globulins, nucleoproteins, and a characteristic albuminoid called neurokeratin.

Lipids of nerve tissue: Over one half (51-54%) of the solid content of the nerve tissue is lipid material. In fact, this tissue is one of the highest in lipid content. (Harper & Rodwell, 1977) [4]

Neuronal Transport Mechanisms

Proteins, organelles, and other cellular materials must be transported throughout the neuron for the maintenance of structural integrity and cellular function. The neuron has transport mechanisms for moving cellular components in an anterograde direction, away from the soma, or in a retrograde direction, toward the soma. The microtubule- associated protein (MAP) kinesin is the anterograde molecular motor that moves organelles and vesicles from the minus to the plus ends of the microtubules via the hydrolysis of adenosine triphosphate. The MAP dynein is the retrograde motor.

Anterograde transport: *Anterograde transport* in the axon occurs at both slow and fast rates. The rate of slow axoplasmic transport is 1 to2 mm/d. Structural proteins, such as actin, neuro-filaments, and microtubules, are transported at this speed. Slow axonal transport is rate limiting for the regeneration of axons following injury to the neuron. The rate of fast axoplasmic transport is about 400 mm/d. Fast transport mechanisms are used for organelles, vesicles, and membrane glycoproteins needed at the axon terminal. In dendrites, anterograde transport occurs at a rate of approximately 0.4 mm/d.

Retrograde transport: Retrograde transport is the process by which material is moved from terminal endings back to the cell body. This process also provides a mechanism for the cell body to sample the environment around its synaptic terminals. In some neurons, maintenance of synaptic connections depends on the trans-neuronal transport of trophic substances such as nerve growth factor, across the synapse from target cells. After retrograde transport to the soma, nerve growth factor activates mechanisms for protein synthesis. Some neurodegenerative disorders may reflect loss of trophic substance delivery as a result of a defect in axonal transport machinery.

Some of the toxic effects of cancer chemotherapy occur secondary to impairment of axoplasmic transport. (Rhoades & Bell, 2013) Neurons display great diversity in size and shape. For example, their cell bodies range in diameter from 5 micro-meters (slightly smaller than a red blood cell) up to 135 *micro*-metre (barely large enough to see with the unaided eye). The pattern of dendritic branching is varied and distinctive for neurons in different parts of the nervous system. A few small neurons lack an axon, and many others have very short axons. As we have already discussed, the longest axons are almost as long as a person is tall, extending from the toes to the lowest part of the brain.

Functional Classification

Functionally, neurons are classified according to the direction in which the nerve impulse (action potential) is conveyed with respect to the CNS.

1. Sensory or afferent neurons (*af*-toward; *-ferrent*-carried) either contain sensory receptors at their distal ends (dendrites) or are located just after sensory receptors that are separate cells. Once an appropriate stimulus activates a sensory receptor, the sensory neuron forms an action potential in its axon and the action potential is conveyed *into* the CNS through cranial or spinal nerves. Most sensory neurons are unipolar in structure.
2. Motor or efferent neurons (*ef*- away from) convey action potentials *away* from the CNS to effectors (muscles and glands) in the periphery (PNS) through cranial or spinal nerves Motor neurons are multipolar in structure.
3. Interneurons or association neurons are mainly located within the CNS between sensory and motor neurons. Interneurons integrate (process) incoming sensory information from sensory neurons and then elicit a motor response by activating the appropriate motor neurons. Most interneurons are multipolar in structure. (Tortora & Derrickson, 2012) [7]

Neurilemma and Myelin Sheath

All axons in the PNS (myelinated and unmyelinated) are surrounded by a continuous living sheath of Schwann cells, known as the neurilemma, or sheath of Schwann. The axons of the CNS, by contrast, lack a neurilemma (Schwann cells are found only in the PNS). This is significant in terms of regeneration of damaged axons, as will be described shortly. Some axons in the PNS and CNS are surrounded by a myelin sheath. In the PNS, this insulating covering is formed by successive wrappings of the cell membrane of Schwann cells; in the CNS, it is formed by oligodendrocytes. Those axons smaller than 2 micrometers (2 μ m) in diameter are usually *unmyelinated* (have no myelin sheath), whereas those that are larger are likely to be *myelinated.*

Myelinated axons conduct impulses more rapidly than those that are unmyelinated. Myelin Sheath in PNS In the process of myelin formation in the PNS, Schwann cells roll around the axon, much like a roll of electrician's tape is wrapped around a wire. Unlike electrician's tape, however, the Schwann cell wrappings are made in the same spot, so that each wrapping overlaps the previous layers. The number of times the Schwann cells wrap themselves around the axon, and thus the number of layers in the myelin sheath, is greater for thicker than for thinner axons. The cytoplasm, meanwhile, is forced into the outer region of the Schwann cell, much as toothpaste is squeezed to the top of the tube as the bottom is rolled up. Each Schwann cell wraps only about a millimeter of axon, leaving gaps of exposed axon between the adjacent Schwann cells. These gaps in the myelin sheath are known as the nodes of Ranvier. The successive wrappings of Schwann cell membrane provide insulation around the axon, leaving only the nodes of Ranvier exposed to produce nerve impulses. The Schwann cells remain alive as their cytoplasm is forced to the outside of the myelin sheath. As a result, myelinated axons of the PNS are surrounded by a living sheath of Schwann cells, or neurilemma. Unmyelinated axons are also surrounded by a neurilemma, but they differ from myelinated axons in that they lack the multiple wrappings of Schwann cell plasma membrane that compose the myelin sheath. Myelin Sheath in CNS As mentioned earlier, the myelin sheaths of the CNS are formed by oligodendrocytes. This

process occurs mostly postnatally (after birth). Unlike a Schwann cell, which forms a myelin sheath around only one axon, each moligodendrocyte has extensions, like the tentacles of an octopus, that form myelin sheaths around several axons. The myelin sheaths around axons of the CNS give this tissue a white color; areas of the CNS that contain a high concentration of axons thus form the white matter. The gray matter of the CNS is composed of high concentrations of cell bodies and dendrites, which lack myelin sheaths. (Fox, 2011) [2]

Electrical Signals in Neuron

Like muscle fibres, neurons are electrically excitable. They communicate with one another using two types of electrical signals: (1) Graded potentials are used for short distance communication only. (2) Action potentials allow communication over long distances within the body. Recall that an action potential in a muscle fibre is called a muscle action potential. When an action potential occurs in a neuron (nerve cell), it is called a nerve action potential (nerve impulse). The production of graded potentials and action potentials depends on two basic features of the plasma membrane of excitable cells: the existence of a resting membrane potential and the presence of specific types of ion channels. Like most other cells in the body, the plasma membrane of excitable cells exhibits a membrane potential, an electrical potential difference (voltage) across the membrane. In excitable cells, this voltage is termed the resting membrane potential. The membrane potential is like voltage stored in a battery. If you connect the positive and negative terminals of a battery with a piece of wire, electrons will flow along the wire. This flow of charged particles is called current. In living cells, the flow of ions (rather than electrons) constitutes the electrical current. Graded potentials and action potentials occur because the membranes of neurons contain many different kinds of ion channels that open or close in response to specific stimuli. Because the lipid bilayer of the plasma membrane is a good electrical insulator, the main paths for current to flow across the membrane are through the ion channels. (Tortora & Derrickson, 2009) An increase in membrane permeability to a specific ion results in the diffusion of that ion down its *electrochemical gradient* (concentration and electrical gradients, considered together), either into or out of the cell. These *ion currents* occur only across limited patches of membrane where specific ion channels are located. Changes in the potential difference across the membrane at these points can be measured by the voltage developed between two microelectrodes (less than 1μm in diameter)—one placed inside the cell and the other placed outside the plasma membrane at the region being recorded. The voltage between these two recording electrodes can be visualized by connecting them to a computer or oscilloscope. All cells have a resting membrane potential, but its magnitude can be different in different types of cells. Neurons maintain an average rmp of − 70 mV, for example, whereas heart muscle cells may have an rmp of − 85 mV. If appropriate stimulation causes positive charges to flow into the cell, the line will deflect upward. This change is called depolarization (or *hypo polarization*) because the potential difference between the two recording electrodes is reduced. A return to the resting membrane potential is known as repolarization. If stimulation causes the inside of the cell to become more negative than the resting membrane potential, the line on the oscilloscope will deflect downward. This change is called hyperpolarization. Hyperpolarization can be caused either by positive charges leaving the cell or by negative charges entering the cell. Depolarization of a dendrite or cell body is *excitatory*, whereas hyperpolarization is *inhibitory*, in terms of their effects on the production of nerve impulses. The reasons for this relate to the nature of nerve impulses (action potentials). (Fox, 2011) [2]

Factors That Affect the Speed of Propagation

The speed of propagation of an action potential is affected by three major factors: amount of myelination, axon diameter, and temperature.

1. **Amount of myelination:** As you have just learned, action potentials propagate more rapidly along myelinated axons than along unmyelinated axons
2. **Axonal diameter:** Larger-diameter axons propagate action potentials faster than smaller ones due to their larger surface areas.
3. **Temperature:** Axons propagate action potentials at lower speeds when cooled. (Tortora & Derrickson, 2012) [7]

Direction of Propagation

An excitable membrane has no single direction of propagation, but the action potential travels in all directions away from the stimulus—even along all branches of a nerve fiber—until the entire membrane has become depolarized.

All-or-None Principle

Once an action potential has been elicited at any point on the membrane of a normal fibre, the depolarization process travels over the entire membrane if conditions are right, or it does not travel at all if conditions are not right. This is called the *all-ornothing principle*, and it applies to all normal excitable tissues. Occasionally, the action potential reaches a point on the membrane at which it does not generate sufficient voltage to stimulate the next area of the membrane. When this occurs, the spread of depolarization stops. Therefore, for continued propagation of an impulse to occur, the ratio of action potential to threshold for excitation must at all times be greater than 1. This "greater than 1" requirement is called the *safety factor* for propagation. (Guyton & Hall, 2006)

Continuous and Saltatory Conduction: There are two types of propagation: continuous conduction and saltatory conduction. The type of action potential propagation described so far is continuous conduction, which involves step-by-step depolarization and repolarization of each adjacent segment of the plasma membrane. In continuous conduction, ions flow through their voltage-gated channels in each adjacent segment of the membrane. Note that the action potential propagates only a relatively short distance in a few milliseconds. Continuous conduction occurs in unmyelinated axons and in muscle fibers. Action potentials propagate more rapidly along myelinated axons than along unmyelinated axons. If you compare parts a and b in you will see that the action potential propagates much farther along the myelinated axon in the same period of time. Saltatory conduction (*saltat-*_leaping), the special mode of action potential propagation that occurs along myelinated axons, occurs because of the uneven distribution of voltage-gated channels. Few voltage-gated channels are present in regions where a myelin sheath covers the axolemma. By contrast, at the nodes of Ranvier (where there is no myelin sheath), the axolemma has many voltage-gated channels. Hence, current carried by Na+ and K+ flows across the membrane mainly at the nodes. When an

action potential propagates along a myelinated axon, an electric current (carried by ions) flows through the extracellular fluid surrounding the myelin sheath and through the cytosol from one node to the next. The action potential at the first node generates ionic currents in the cytosol and extracellular fluid that depolarize the membrane to threshold, opening voltage-gated Na+ channels at the second node. The resulting ionic flow through the opened channels constitutes an action potential at the second node.

Then, the action potential at the second node generates an ionic current that opens voltage-gated Na+ ion channels at the third node, and so on. Each node repolarizes after it depolarizes. The flow of current across the membrane only at the nodes of Ranvier has two consequences:

1. The action potential appears to "leap" from node to node as each nodal area depolarizes to threshold, thus the name "saltatory." Because an action potential leaps across long segments of the myelinated axolemma as current flows from one node to the next, it travels much faster than it would in an unmyelinated axon of the same diameter.
2. Opening a smaller number of channels only at the nodes, rather than many channels in each adjacent segment of membrane, represents a more energy-efficient mode of conduction. Because only small regions of the membrane depolarize and repolarize, minimal inflow of Na_ and outflow of K_ occurs each time an action potential passes by. Thus, less ATP is used by sodium–potassium pumps to maintain the low intracellular concentration of Na_ and the low extracellular concentration of K+. (Tortora & Derrickson, 2012) [7]

Nerve Conduction Study

In the past two decades, major advances have taken place in the field of peripheral nerves, Especially, in relation to its ultra-structure, histo-chemistry, neuro-physiology and axonal transport system. These advances have not only contributed to a better understanding of normal peripheral nerve structure and function but also in relation to various diseases. (Misra, 2012) [5]

Nerve conduction study is done to assess whether a nerve which has suffered compression or injury is degenerating or not. The nerve is stimulated directly by a short duration stimulus along its course. When it is stimulated it conveys impulses to the muscles it supplies and the muscles contract (Downie, 1992) [1]. The principal use of nerve conduction studies is to identify damage to peripheral nerves, and to determine whether the pathological process is focal or diffuse and whether the damage is principally axonal or demyelinating. It is also possible to obtain some information about nerve roots by more sophisticated analysis of responses to impulses initially conducted antidromically to the spinal cord, and then orthodromically to the stimulation point (F wave). (Walker *et al*., 2013) [8]

Nerve Conduction Velocity

By stimulating a motor nerve at two different points along its course and by recording from an appropriate muscle the motor unit potentials so produced, it is possible to measure the stimulus contraction delay interval in each case and hence to calculate the rate of conduction of the impulse along the nerve. (Walton, 1979) [9]

Principle of Motor Nerve Conduction Study

The motor or mixed nerve is stimulated at least at two points along its course. The pulse is adjusted to record a compound muscle action potential. It is important to ensure a supra-maximal stimulation keeping the cathode close to the active recording electrode. This prevents hyper polarisation effect of anode and anodal conduction block. The surface recording electrodes are commonly used and placed in belly tendon montage keeping the active electrode close to the motor point and reference to the tendon. Ground electrode is placed between stimulating and recording electrodes. A biphasic action potential with initial negativity is thus recorded. Surface stimulation of healthy nerve requires a square wave pulse of 0.1 ms duration with an intensity of 5-40 mA. In a diseased nerve, however, the nerve excitability is reduced and the current requirement may be much higher than the normal. Filter setting for motor nerve conduction study is 5 Hz to 10 KHz and sweep speed 2-5 ms/division. The measurement for motor nerve conduction study include the onset latency, duration, amplitude of compound muscle action potential (CMAP) and nerve conduction velocity. The onset latency is the time in milliseconds from the stimulus artifacts to the first negative deflection of CMAP. For better visualization of the take-off, the latency should be measured at a higher gain than the one used for CMAP amplitude measurement. The onset latency is a measure of conduction in the fastest conducting motor fibers. It also includes neuromuscular transmission time and the propagation time along the muscle membrane which constitute the residual latency. The amplitude of CMAP is measured from baseline to the negative peak (base to peak) or between negative and positive peaks (peak to peak). The amplitude correlates with the number of nerve fibres. The duration of CMAP is measured from the onset to the negative or positive peak or the final return of wave form to the base line. Duration correlates with the density of small fibres. The area under the CMAP can also be measured. However, it needs computer analysis. Motor nerve conduction velocity is calculated measuring the distance between two points of stimulation in mm which is divided by the latency difference in millisecond. The nerve conduction velocity is expressed as m/s. Measurement of latency difference between the two points of stimulation eliminates the effect of residual latency.

Conduction Velocity = D / (Pl-Dl) M/S

PL= Proximal latency in ms

DL= Distal latency in ms

D= Distance between proximal and distal stimulation in mm.

For accurate motor nerve conduction velocity measurements, the distance between two points of stimulation should be at least 10cm. (Misra, 2012) [5].

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